Spring 2018

The NCVDLS REPORT

Accredited by the American Association of Veterinary Laboratory Diagnosticians

Veterinary News and Information From North Carolina's Diagnostic Laboratories

A Message from Our Director...

North Carolina Department of Agriculture and Consumer Services

Steve Troxler, Commissioner

The Rollins Diagnostic Laboratory was happy to assist students and poultry industry veterinarians by hosting a training lab in our necropsy room this spring. NCSU's Poultry Diseases course had a record number of students enroll, and they were having difficulty finding a necropsy facility to house the training/teaching laboratory. We were glad to step in and lend a hand by letting them use our facility and provide assistance during the training. Colleagues from the Veterinary College, Poultry Industry, and Diagnostic lab worked together to make this a great learning experience for the students!

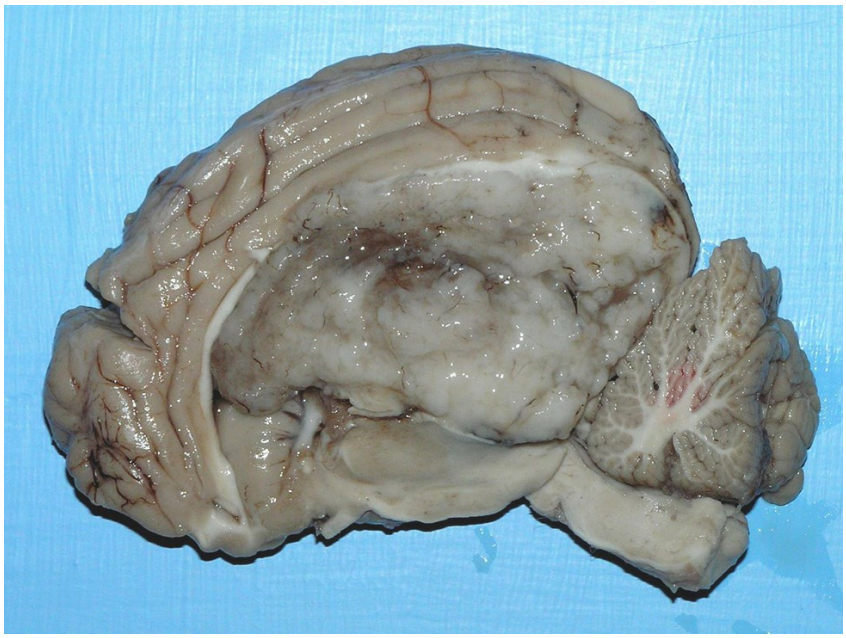
Jim Trybus, DVM, DAVCP

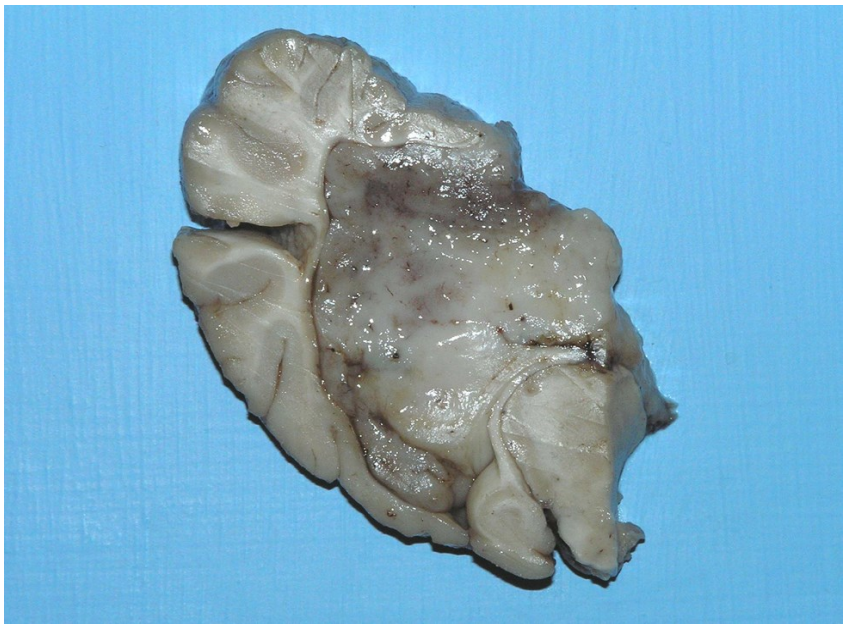
Neurologic Disease in an Alpaca

By: Steven Rushton DVM, Diplomate ACVP

Presented to the North Carolina Veterinary Diagnostic Laboratory was a 2 year old, female, intact Alpaca. The animal had a six-day history of neurologic signs that included head tilt, a dysmetric gait and blindness. Treatment by the referring veterinarian included Fenbendazole for possible Parelaphostrongylus migration, antibiotics for possible Listeriosis and Thiamine for possible Polioencephalomalacia.

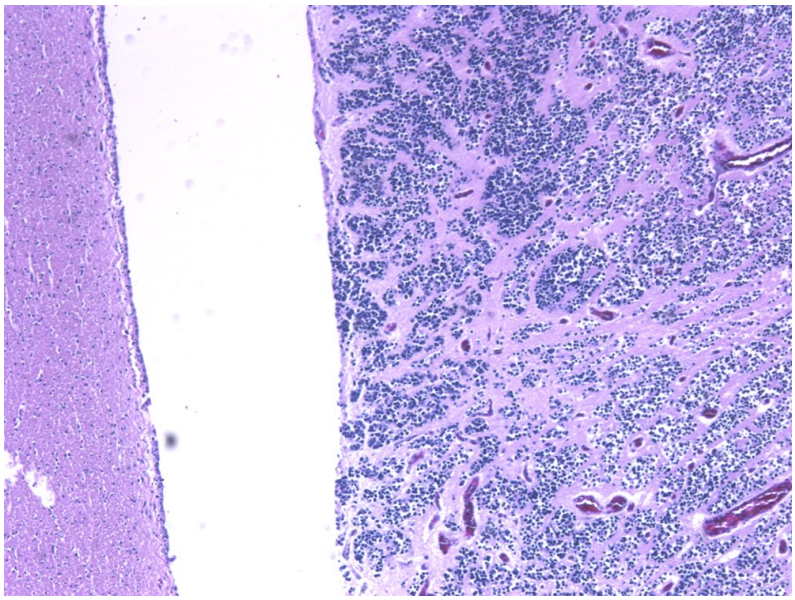
The approximately 80 kg alpaca is in good body condition and mildly dehydrated. There was a tan, 8 x 9cm slightly lobulated mass located in the left lateral ventricle extending through the corpus callosum filling the adjacent right ventricular space. This mass moderately compresses the surrounding cerebral parenchyma. Mild cerebellar herniation through the foramen magnum was also identified. No other gross lesions are identified.

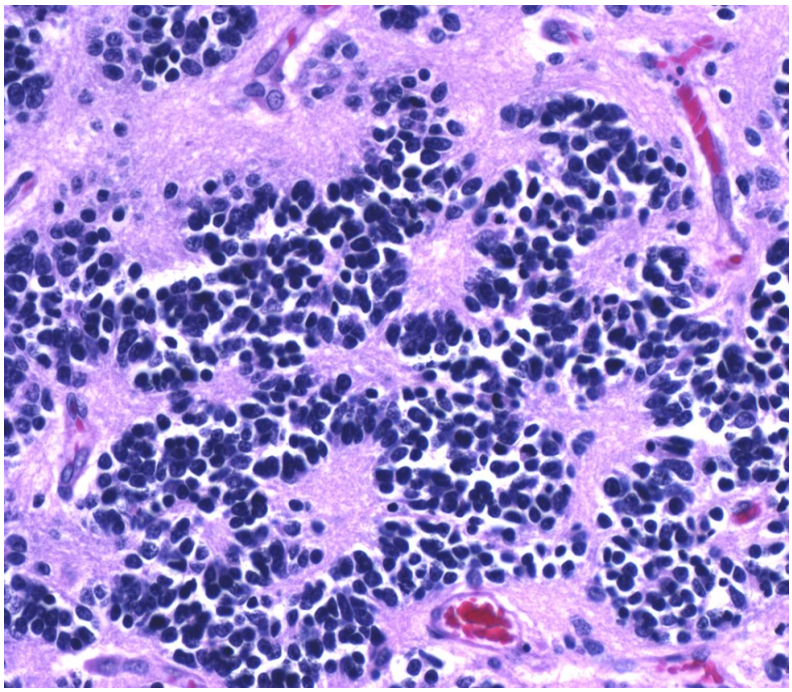




Histologically, compressing the adjacent cortical neuropil is a densely cellular, well-circumscribed, unencapsulated neoplasm composed of polygonal cells arranged in solidly cellular areas, true rosettes (both Flexner-Wintersteiner and Homer Wright variants), and pseudorosettes on a fine fibrovascular stroma. Neoplastic cells have indistinct cell borders, small to moderate amounts of eosinophilic, fibrillar to granular cytoplasm, and, rare, poorly discernible, pale luminal cilia. Nuclei are round to oval and basally oriented with dense to coarsely stippled chromatin. There is minimal anisocytosis and anisokaryosis and a mitotic rate of 1 per 10 HPF. Within the neoplasm there are multifocal areas of eosinophilic cellular and karyorrhectic debris (necrosis), fibrin and hemorrhage. Within the adjacent neuropil, there is mild lymphocytic perivascular cuffing of blood vessels which occasionally have a reactive endothelial lining, increased numbers of glial cells (gliosis), and, rarely at the interface with the neoplasm, moderate numbers of gitter cells.

Diagnosis: Neuroectodermal Tumor suggestive of Ependymoma





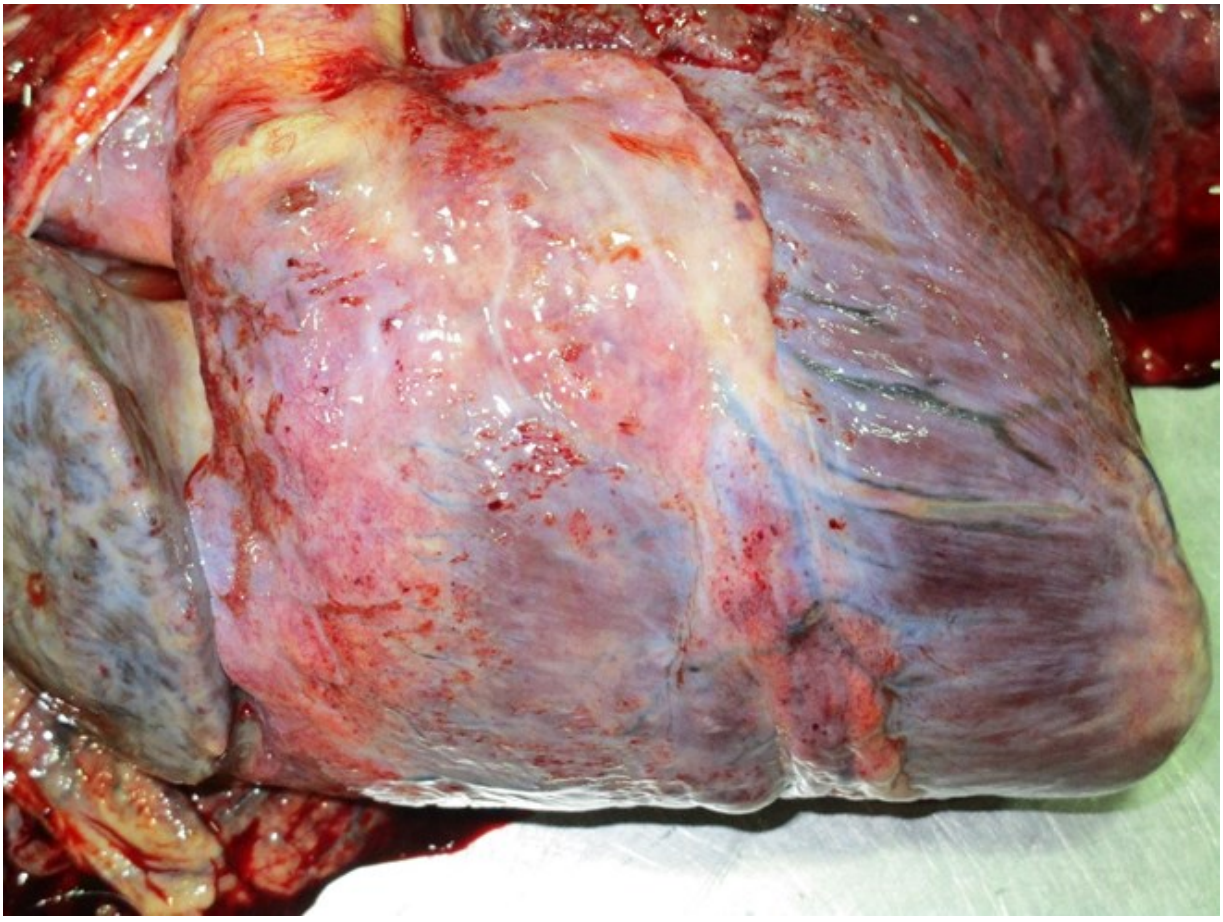
Immunohistochemistry was performed to help further characterize the neoplastic cells. Neuroendocrine tumor marker Synaptophysin was negative. Neuroectodermal marker GFAP showed positively staining cells. The immunohistochemistry results were consistent with a Neuroectodermal tumor and considering the location the final diagnosis was an Anaplastic Ependymoma.

Ependymomas are tumors that arise from the ependymal cells lining the ventricles and central canal of the spinal cord and can arise from the lateral, third, and fourth ventricles, mesencephalic aqueduct or central canal. Ependymomas are reported in nonhuman primates, dogs, cats, rats, cattle, horses, deer, and fish; more prevalent in dogs. At the time of the necropsy the only reported brain tumor in alpacas was an Astrocytoma.

Malignant Catarrhal Fever at a Zoological Park

By: Dr. Jessica Kees

A 46kg, 4-year old, female, Axis deer from a multispecies zoological facility in very thin body condition was submitted to the laboratory for necropsy with a brief history of recumbency and distress observed earlier that morning. On examination, the ocular sclera was yellow and the corneas cloudy and opaque. The epicardium of the right ventricle was roughened and irregular and paintbrush hemorrhages were present especially along the coronary and interventricular grooves. Pale tan foci were scattered randomly throughout the myocardium. An enhanced reticular pattern was observed in the liver and a few to several, random, flat, pale tan foci, 2-5mm in diameter were scattered throughout the parenchyma. The urinary bladder contained reddish brown urine. A urine dipstick showed the urine had a specific gravity of 1.025, pH of 6, bilirubinuria, proteinuria, and hematuria.



The following day, a 113kg, 3-year old, female, water buck antelope from the same zoological facility was submitted for necropsy with a four-day history of bloody diarrhea, inappetence, and fever. The animal was in emaciated body condition. Over 1000ml of serous to serosanguinous fluid was present in the abdominal cavity, 15-20ml of serosanguinous fluid was present in the pericardium, and 20-25ml of serosanguinous fluid was present in the thoracic cavity. Paintbrush to suffusive hemorrhages were present throughout the endocardium of the right and left ventricle. The abomasum was filled with dark green to brown liquid, black gritty sand-like material, numerous small pebbles and rocks and many, often coalescing, 1-2mm diameter to 1-2mm linear, depressed erosions were present throughout the mucosa.

On histopathological examination, in addition to fibrinous vascular necrosis observed in many organs, a lymphoplasmacytic and neutrophilic cystitis, steatitis, nephritis, myocarditis, and hepatitis was also present in the deer. A lymphocytic and necrotizing vasculitis accompanying a lymphocytic cystitis, myocarditis, and interstitial nephritis was observed histologically in the antelope. An erosive, neutrophilic and lymphocytic abomasitis was also observed in this animal.

The lymphocytic vasculitis, perivascular inflammation, and the accumulation of lymphoid cells in nonlymphoid tissues were consistent with Malignant Catarrhal Fever (MCF) and tissues submitted from both the deer and the antelope for PCR testing at the Washington Animal Disease Diagnostic Laboratory were positive for ovine herpesvirus-2, confirming the diagnosis. Ovine herpesvirus-2 is the main cause of MCF in domesticated animals outside of Africa.

MCF is a sporadic, often fatal, multisystemic disease that affects cattle, small ruminants, and wild ruminants. MCF is caused by a gammaherpesvirus and primarily affects lymphoid tissues, epithelial tissues (especially the respiratory system and intestinal tract) and the vasculature, causing lymphoproliferative lesions, vasculitis, and erosive to ulcerative cutaneous and mucosal lesions. There are multiple MCF viruses and each virus is highly adapted to one host species. Sheep and wildebeest are the primary carriers of the virus and while the host animals are usually asymptomatic, they can spread the virus and cause disease in other species. Most domestic sheep are thought to be infected with ovine herpesvirus-2. The virus is excreted mainly in ocular and nasal secretions and inhalation is thought to the primary route of infection however vertical transmission (in utero) is possible. Animals become infected when they come in close contact with sheep or wildebeest. MCF can cause a variety of clinical signs in the different species. High fever, inappetence, and corneal edema are common signs seen in cattle. Depression, diarrhea, and weakness can also occur. The same clinical signs can occur in antelope but they may have less severe and less specific lesions than cows. In deer, MCF often causes peracute disease and death occurs within only a few days so clinical signs may not be observed. In some outbreaks that occur in deer, typical clinical signs observed include: depression, loss of condition, a rough hair coat, corneal opacity, hemorrhagic diarrhea, and bloody urine. There is no specific antiviral therapy available and treatment is typically aimed at controlling secondary infections and supportive care.

The animals submitted for necropsy came from a zoological park where sheep and susceptible species such as deer and antelope co-mingle. Despite this not being an issue in the past at this particular park, the risk of housing other ruminant species with sheep is great and therefore co-mingling should be avoided wherever possible.

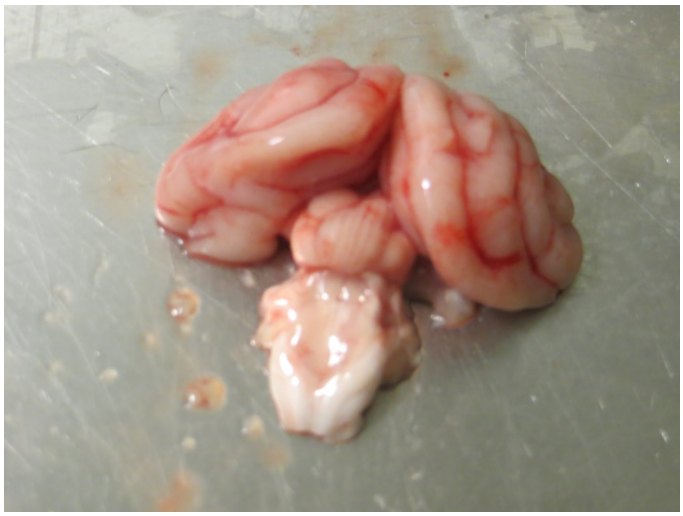
NCVDLS has diagnosed Canine Distemper Virus in 4 dogs from Anson County, one raccoon from Union County, and one fox from Moore County in March and April 2018. Veterinarians in Anson, Union, and Moore Counties and the surrounding areas should consider Canine Distemper Virus as a possible differential diagnosis for any unvaccinated dog or puppy presenting with upper respiratory or neurological signs.

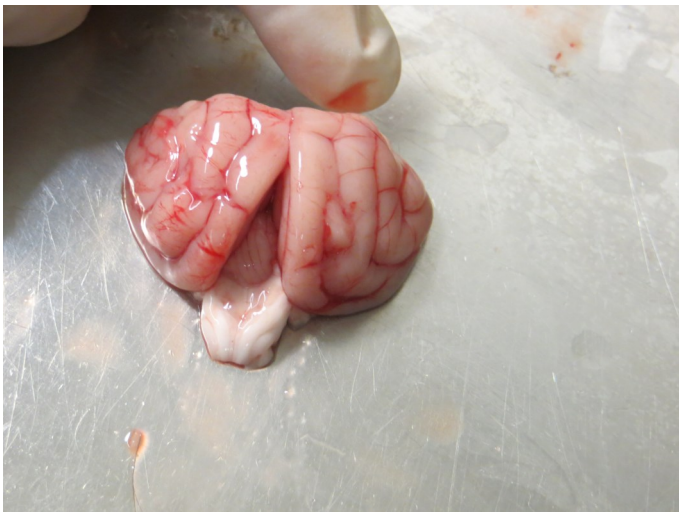
Cerebellar Hypoplasia

By: Dr. Heather Wyss

A good Samaritan found 5, eight week old feral kittens. Two of the kitten were brought to her veterinarian for displaying neurological signs. The kittens were ataxic, tremoring, and had altered mentation. The kitten presented for necropsy bit someone during handling. A necropsy and rabies testing were requested.

The necropsy examination revealed a severely underdeveloped cerebellum. The remainder of the necropsy examination was unremarkable with the exception of fleas.





Cerebellar hypoplasia usually occurs when a pregnant female contracts feline panleukopenia during gestation. The parvovirus attacks actively dividing cells. As the virus infects the rapidly dividing cells of the cerebellum, it causes a degeneration of granule and Purkinje cells and interferes with cortical development, resulting in cerebellar hypoplasia. Kittens may be stillborn or die shortly after birth. In surviving kittens, the severity of the hypoplasia can vary among littermates and range from mild to severe. The signs are often noticeable when they start to ambulate. Clinical signs include ataxia, intention tremors, a broad based stance, and hypermetria.

The clinical signs of cerebellar hypoplasia do not progress or worsen over time. However, some cats will learn to compensate giving the appearance of improvement.

Modified live versions of the panleukopenia vaccine can also cause cerebellar hypoplasia if administered later in gestation. The time of most concern is the last trimester. Vaccines should be withheld from nursing queens until the kittens are three to four weeks old as cerebellar development continues during the first two weeks after birth.

In the absence of other congenital or health issues, cats with cerebellar hypoplasia have a normal life expectancy.

Cats with cerebellar hypoplasia should be kept strictly indoors to help avoid injury.

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